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Lipoprotein implication and Laboratory estimation

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Abstract

Cholesterol, triglycerides, and high-density lipoproteins are important constituents of the lipid fraction of the human body. Cholesterol is an unsaturated alcohol of the steroid family of compounds; it is essential for the normal function of all animal cells and is a fundamental element of their cell membranes. A lipoprotein is a biochemical assembly that contains both proteins, and lipids, bound to the proteins, which allow fats to move through the water inside and outside cells. Plasma lipoproteins are separated by hydrated density; electrophretic mobility; size; and their relative content of cholesterol, triglycerides, and protein into five major classes: chylomicrons, very-low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). The role of lipoprotein particles is to transport triacylglycerols and cholesterol in the blood between all the tissues of the body; the most common being the liver and the adipocytes of the adipose tissue. The liver is the central platform for the handling of lipids: it is able to store glycerols and fats in its cells, the hepatocytes. Hepatocytes are also able to create triacylglycerols via de novo synthesis. And they also produce the bile from cholesterol. Lipids have important roles in virtually all aspects of life, ranging from serving as hormones, energy source, aiding in digestion, to acting as structural components of the membranes of cells. All these good attributes do not undermine the fact that lipids are involved in the development of atherosclerosis, a pathogenic process that is the underlying cause of the common cardiovascular disorders such as, myocardial infarction, cerebrovascular disorders, and other peripheral vascular diseases. In other to ensure that these anomalies' are curtail, lipid has to be maintained physiologically, evenly distributed and transported in the body system which are the primary function of lipoproteins.

Keywords: Lipoproteins, Lipoprotein implications, Laboartory Estimation

Introduction

Cholesterol, triglycerides, and high-density lipoproteins are important constituents of the lipid fraction of the human body. Cholesterol is an unsaturated alcohol of the steroid family of compounds; it is essential for the normal function of all animal cells and is a fundamental element of their cell membranes. It is also a precursor of various critical substances such as adrenal and gonadal steroid hormones and bile acids. Triglycerides are fatty acid esters of glycerol and represent the main lipid component of dietary fat and fat depots of animals.

Cholesteryl esters and triglycerides, being non-polar lipid substances, need to be transported in the plasma associated with various lipoprotein particles (AHA,1984).

A lipoprotein is a biochemical assembly that contains both proteins, and lipids, bound to the proteins, which allow fats to move through the water inside and outside cells. The protein molecules are called apoproteins and serve to emulsify the lipid molecules (AHA,1984). Plasma lipoproteins are separated by

hydrated density; electrophretic mobility; size; and their relative content of cholesterol, triglycerides, and protein into five major classes: chylomicrons, very-low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL) (Burtis *et al.*,2008).

The role of lipoprotein particles is to transport triacylglycerols and cholesterol in the blood between all the tissues of the body; the most common being the liver and the adipocytes of the adipose tissue. Particles are synthesized in the small intestine and the liver, but interestingly not in the adipocytes (AHA,1984). The terms "good" and "bad" cholesterol erroneously refer to High Density Lipoproteins (HDL) and Low Density Lipoproteins (LDL), respectively. They are estimated in the Laboratories. High levels of LDL are associated with coronary atherosclerosis, whereas high levels of HDL appear to protect against cardiovascular diseases.

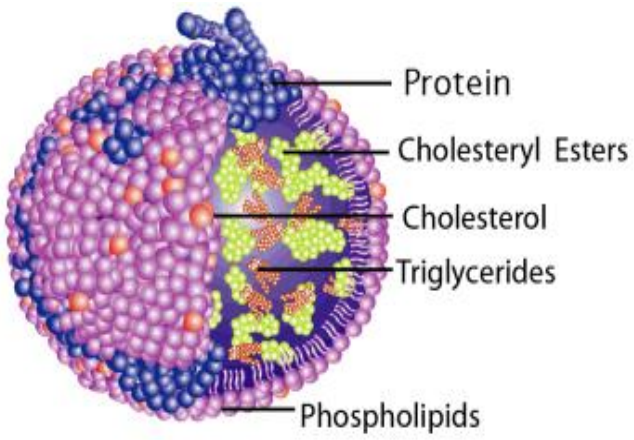
Plasma lipoprotein particles

Lipids synthesized in the liver and intestine are transported in the plasma in macromolecular complexes known as lipoproteins.

Chemistry

Plasma Lipoproteins are small spherules that transport fats in the body and consist of protein, cholesterol, triglycerides, and phospholipids. The lipoprotein particles have hydrophilic, polar amphipathic groups of phospholipids and apoproteins directed outward. Such characteristics make them soluble in the salt water-based blood pool. Triglyceride-fats and cholesteryl esters are carried internally, shielded from the water by the phospholipid monolayer and the apoproteins (Apo A, B, C and E).The association of the core lipids with the phospholipids and apoproteins is non covalent, occurring primarily through hydrogen bonding and van der waals forces (Burtis *et al.*,2008). The binding of lipids and apoproteins is weak and allows their exchange among the lipoproteins and between cell membranes and lipoproteins. The binding is sufficiently strong, however, to allow the various classes of lipoprotein to be isolated by a variety of analytical techniques (Burtis *et al.*,2008).

The interaction of the proteins forming the surface of the particles (a) with enzymes in the blood, (b) with each other, and (c) with specific proteins on the surfaces of cells determine whether triglycerides and cholesterol will be added to or removed from the lipoprotein transport particles (Brewer *et al.*,1983).



Structure of lipoprotein particle.

Metabolism of lipoprotein particles

The handling of lipoprotein particles in the body is referred to as lipoprotein particle metabolism. It is divided into two pathways, exogenous and endogenous, depending in large part, on whether the lipoprotein particles in question are composed chiefly of dietary (exogenous) lipids or whether they originated in the liver (endogenous), through de novo synthesis of triacylglycerols.

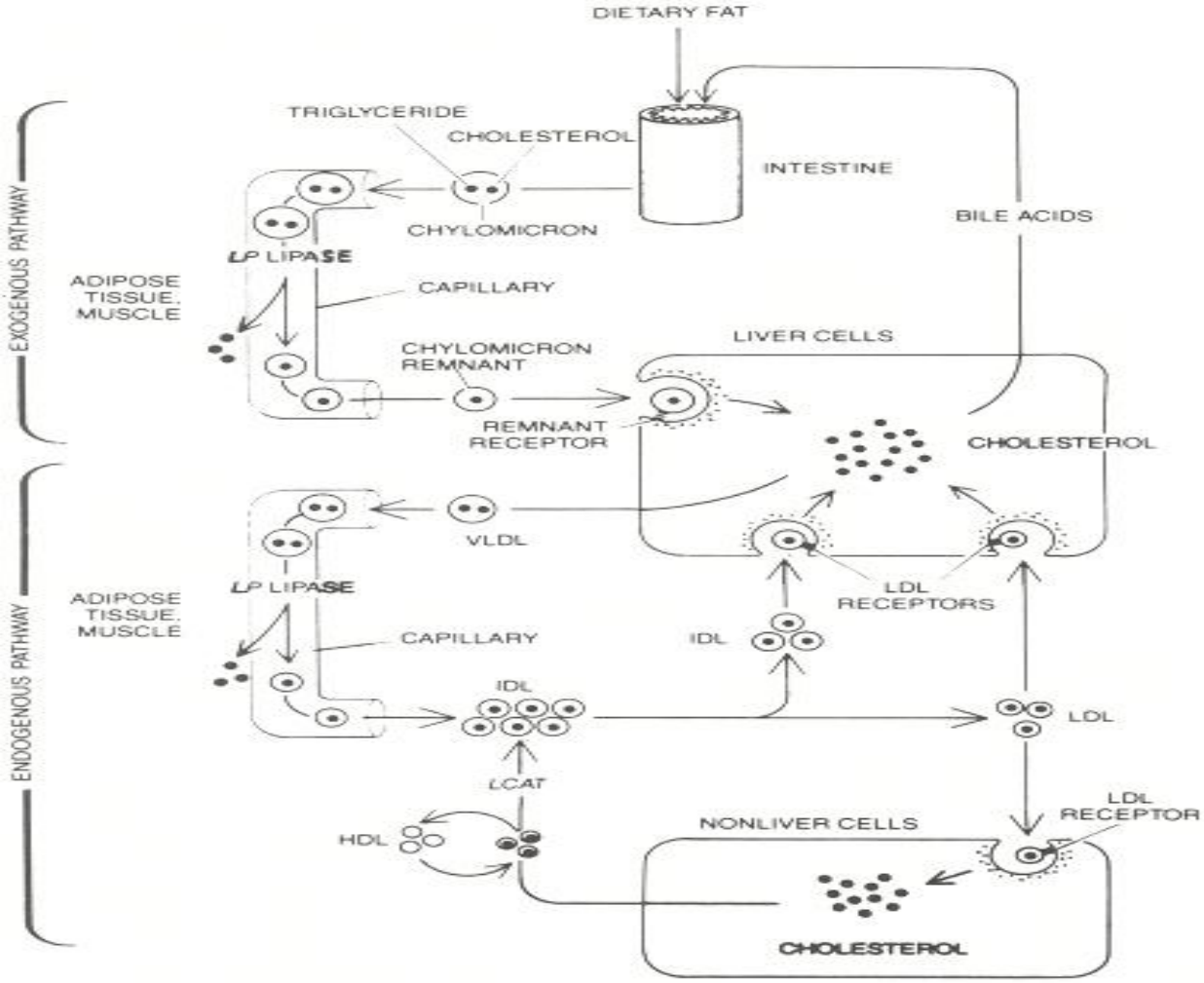
The hepatocytes are the main platform for the handling of TGs and cholesterol; the liver can also store certain amounts of glycogen and triacylglycerols. Intriguingly, adipocytes, though being the main storage cells for triacylglycerols, do not produce any kind of lipoprotein particle (AHA,1984).

Exogeneous pathway

Bile emulsifies fats contained in the chyme, and then pancreatic lipase cleaves triacylglyceride molecules into two fatty acids and one 2-monoacylglycerol. Enterocytes readily absorb these molecules from the chymus. Inside of the enterocytes, fatty acids and monoacylglycerides are transformed again into triacylglycerides. Then these lipids (triacylglycerols, phospholipids, cholesterol, and cholesteryl esters) are assembled with apolipoprotein B-48 into nascent chylomicrons. These particles are then secreted into the lacteals in a process that depends heavily on apolipoprotein B-48. As they circulate through the lymphatic vessels, nascent chylomicrons bypass the liver circulation and are drained via the thoracic duct into the bloodstream.

In the blood stream, nascent chylomicron particles bump with HDL particles; as a result, HDL particles donate apolipoprotein C-II and apolipoprotein E to the nascent chylomicron; the chylomicron is now considered mature. Via apolipoprotein C-II, mature chylomicrons activate lipoprotein lipase (LPL), an enzyme on endothelial cells lining the blood vessels. LPL catalyzes the hydrolysis of triacylglycerol (glycerol covalently joined to three fatty acids) that ultimately releases glycerol and fatty acids from the chylomicrons. Glycerol and fatty acids can then be absorbed in peripheral tissues, especially adipose and muscle, for energy and storage.

The hydrolyzed chylomicrons are now called chylomicron remnants. The chylomicron remnants continue circulating the bloodstream until they interact via apolipoprotein E with chylomicron remnant receptors, found chiefly in the liver. This interaction causes the endocytosis of the chylomicron remnants, which are subsequently hydrolyzed within lysosomes. Lysosomal hydrolysis releases glycerol and fatty acids into the cell, which can be used for energy or stored for later use (Craig *et al.*,2000).



Endogeneous pathway

The liver is the central platform for the handling of lipids: it is able to store glycerols and fats in its cells, the hepatocytes. Hepatocytes are also able to create triacylglycerols via de novo synthesis. And they also produce the bile from cholesterol.

In the hepatocytes, triacylglycerols, cholesterol cholesteryl esters are assembled with apolipoprotein B-100 to form nascent VLDL particles. Nascent VLDL particles are released into the bloodstream via a process that depends upon apolipoprotein B-100.

In the blood stream, nascent VLDL particles bump with HDL particles; as a result, HDL particles donate apolipoprotein C-II and apolipoprotein E to the nascent VLDL particle; once loaded with apolipoproteins C-II and E, the nascent VLDL particle is considered mature.

Again like chylomicrons, VLDL particles circulate and encounter LPL expressed on endothelial cells. Apolipoprotein C-II activates LPL, causing hydrolysis of the VLDL particle and the release of glycerol and fatty acids. These products can be absorbed from the blood by peripheral tissues, principally adipose and muscle. The hydrolyzed VLDL particles are now called VLDL remnants or intermediate-density lipoproteins (IDLs). VLDL remnants can circulate and, via an interaction between apolipoprotein E and the remnant receptor, be absorbed by the liver, or they can be further hydrolyzed by hepatic lipase.

Hydrolysis by hepatic lipase releases glycerol and fatty acids, leaving behind IDL remnants, called low-density lipoproteins (LDL), which contain a relatively high cholesterol content (Brewer *et al.*,1983) . LDL circulates and is absorbed by the liver and peripheral cells. Binding of LDL to its target tissue occurs through an interaction between the LDL receptor and apolipoprotein B-100 on the LDL particle. Absorption occurs through endocytosis, and the internalized LDL particles are hydrolyzed within lysosomes, releasing lipids, chiefly cholesterol(Craig *et al.*,2000).

Classification of lipoprotein

They are classified base on their buoyant density, which inversely reflects their size. The greater the lipid to protein ratio, the larger the size and the lesser the density. They can also be classified according to their electrophoretic mobility due to protein content. Those with higher protein content move faster to the anode (Fredrickson's classification)(Burtis *et al.*,2008).

Base on the density, they are classified into five main groups that are important physiologically and in clinical diagnosis. The first three are triglyceride rich and, because of their large size, they scatter light, which can give plasma a turbid appearance (lipaemic), if present in high quantity. The remaining two, which are lesser in size and heavier do not scatter light.

- Chylomicrons are the largest and least dense lipoproteins. They transport exogenous lipids from the intestine to all cells and originate from the gut. They are made up of about 90% triglyceride (Tg), 1% protein (Pro), 4% cholesterol (Chol) and 5% phospholipids (PL). They also contain apoproteins A, B, C and E. Electrophoretically, they appear at the origin. They are just less than 0.95g/ml heavy.
- Very low-density lipoprotein (VLDL) is the next, it transports endogenous lipid from the liver to cells which by such function lend it the chance to be synthesized in the liver. It contains up to 55% of Tg, 25% of Chol, 8% of Pro and 12% of PL. It contains only B, C and E apolipoproteins. It is pre-beta in electrophoretical mobility. Their density range from 0.95-1.006g/ml
- Intermediate – density lipoproteins (IDLs). These ones are known to be transient, formed during the conversion of VLDLs to LDLs. They are normally non-detectable in the plasma and consists of, 9%Chol., 19% PL, 19%Pro., 23%Tg., and of course the sources are VLDL and LDL. They are about 1.006-1.019g/ml heavy. Major proteins here are Apo B-100 and E. They are broad beta in electrophoretic mobility.
- Low – density lipoproteins (LDLs) are formed from VLDLs, IDLs and carry cholesterol from the liver to the cells and tissues, a function that earned them an erroneous name, bad cholesterol. They have the density range of 1.019-1.063g/ml and showcases beta band in electrophoretic mobility. They have such components as,20%Pro., 55% Chol., 5% Tg., 12%PL. They haveApo B-100 as major protein and exhibit beta band electrophoretically.
- High – density lipoproteins are the heaviest amongs the lipoproteins after the Lp(a) with the density of 1.063-1.210g/ml. Their main source being the Gut and the Liver. Their constituents include 50%of Pro, 20% of Chol, 5%Tg, and 25%PL. They can be further divided into HDL_2 and HDL_3; Apos A, C and E are the apoproteins with apos A1 and A2 as the major ones. They showcase alpha band in the electrophoretic mobility. They transport excess cholesterols from the cells to the liver for metabolism and hence, are called good cholesterol,erroneously(Crook,1971).

Lp(a) are distinct class of lipoproteins; they are structurally related to LDL, both containing one molecule of apo B-100 per particle and have similar lipid composition. They contain a carbohydrate-rich protein, apo (a) that is covalently bound to the apo B -100 through a disulphide linkage. They are the densest, with density of 1.040-1.130g/ml and apo (a) and B-100 as their major proteins. They have pre-beta band in their electrophoretic mobility (Burtis *et al.*,2008*)*.

Laboratory analysis of lipoproteins

Routine lipoprotein assay are performed by most clinical laboratories. Corresponding reference methods have been developed by the CDC which have been critical in the standardization of lipoprotein assays (Brewer *et al.*,1983*)*.

Analysis of plasma or serum total cholesterol, triglycerides, and lipoproteins is usually performed on blood specimens obtained by venipuncture in tubes containing EDTA. Because of a larger content of water, plasma levels have been found to be around 3% lower than matched serum levels. Cholesterol levels are fairly constant, but triglyceride levels fluctuate considerably from day to day and are highest 1 to 4 hours after meals (Brewer *et al.*,1983*)*.

Collection of blood for lipoproteins and triglyceride testing should be done after a 12-hour fasting period, when chylomicrons have ordinarily been cleared from the circulation. Measurements must be ideally done while the patients are on their usual diet and taking no medications that could alter blood lipid levels. Sampling should not be performed during periods of stress or within 6 weeks after a major illness, such as an acute myocardial infarction, as plasma cholesterol may be reduced and triglyceride levels increased in these instances.

However, as results from commercial laboratories tend to differ from those obtain from research methods; it is advisable to enquire on how the method used by a particular laboratory compares with the reference value 9Burtis *et al.*,2008*)*.

HDL Analysis

It is a precipitation assay that involves precipitation of non-HDL lipoproteins with a polyionic compound such as heparin, dextran sulfate in the presence of a divalent cation such as Mg^{2+}, Mn^{2+}. Then the cholesterol content is measured Enzymatically.

Method: (cholesterol oxidase peroxidase) or Enzyme Colorimetric Method.

Materials: Test tubes, rack, pipettes and tips, reagent kits, distilled water, water bath, spectrophotometer, centrifuge, cuvet, cotton wool.

Principle: After precipitation with a polyanionic compound, In the presence of water, cholesteryl ester is hydrolyzed by cholesteryl esterase to give cholesterol and fatty acid. The cholesterol formed is further oxidized into 4-cholesten-3-one and H_20_2 by cholesterol oxidase. In the presence of peroxidase, H_20_2 is broken down to release 0_2 which in the presence of phenol, combines with 4-aminoantipyrine to form a red quinine colour complex which is read spectrophotometrically at 500nm.

Procedure:

	Test	Standard
Polyanionic reagent	300µl	300µl
Standard reagent	-	300µl
Sample	300µl	-

Mix and allow to stand for 10mins at room temp.; spin at 4000r.p.m for 10min. Transfer the supernatants into another test tubes as follows:

	Blank	Standard	Sample
Working Rgt.	1ml	1ml	1ml
Supernatants	-	0.5ml	0.5ml
Distilled water	0.5ml	-	-

Mix and incubate at 37℃ in a water bath for 5mins.

Read the absorbance spectrophotometrically at 500nm.[5]

CALCULATION: HDL-C Conc. (mg/dl) = $\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times 200$

Reference values: Males – 35-55mg/dl

Females – 45-65mg/dl

LDL analysis:

Here, both direct and indirect methods can be used. The indirect method assumes that total cholesterol is composed primarily in VLDL, LDL and HDL. Therefore, LDL-Chol. is estimated by the use of Friedwald equation or ß-quantification.[3]

Friedwald equation:

LDL Chol. (mg/dl).= [Total Chol.] – [HDL Chol.] –

([Triglyceride]/5)

ß-QUANTIFICATION: ß-quantification is a tedious procedure and is only reserved for samples in which Friedwald equation is inappropriate, such as those with [Tg] of 400mg/dl; EDTA plasma is a specimen of choice (Burtis *et al.*,2008). The method involves ultracentrifugation of an aliquot of plasma (density =1.006g/ml) at 105,000 for 18hrs at 10℃. Under these

conditions, VLVL, ß-VLDL, Chylomicrons, all accumulate in the floating layer which is removed by tube slicer, while the infranatant with density of greater than 1.006g/ml with mostly LDL and HDL remixed, reconstituted to a known volume, and its cholesterol content measured. The VLDL and LDL Cholesterols are calculated with the following equation

[VLDL Chol.] = [Total Chol.] – [d > 1.006g/ml chol.]

[LDL Chol.] = [d > 1.006g/ml chol.] – [HDL chol.]

Direct measurement of LDL chol. Involves masking of cholesterol associated with other non-LDL fractions or by selectively solubilizing LDL. One early approach took advantage of the fact that apo B-100 is essentially the only apolipoprotein in LDL. A mixture of polyclonal antibodies to apo A~1 and apo E was used to bind and mask cholesterols on VLDL, IDL and HDL, and LDL cholesterol is measured enzymatically (Burtis *et al.*,2008).

Other potable methods have also been developed such as Autoanalyzer (desktop analyzers, physician's office analyzers, point of care analyzers) methods for use in nonlaboratory settings which are capable of accurately and precisely measuring cholesterol and ; most also quantify Tg and HDL cholesterol, with calculation of LDL chol., in a few µl of whole blood, serum, or

plasma within few minutes; which makes these types of analyzers suitable for public cholesterol screening programs (Burtis *et al.*,2008).

Clinical implications and significance

Hyperlipoproteinemia is the lipid disturbance of major relevance clinically because of its association with an increased risk of atherosclerotic cardiovascular disease. Multiple epidemiologic studies have demonstrated that increased levels of plasma total cholesterol and low-density lipoproteins are strongly and directly related to a greater incidence of coronary heart disease. Elevated plasma triglycerides and very-low-density lipoproteins are directly associated with the risk of atherosclerotic heart disease, although not as independent risk factors. In contrast, high levels of high-density lipoprotein cholesterol have been found to be a protective factor for the development of that disease, so that decreased levels constitute a risk factor.

Clinical manifestations of hyperlipoproteinemia include a greater incidence of ischemic vascular disease, acute pancreatitis, and visible accumulations of lipid deposits (xanthomas and xanthelasmas). The localization of these lesions is of great help in many instances to categorize the lipoprotein dysfunction present (Craig *et al.*,2000).

Hyperalphalipoproteinemia

Another clinical condition associated with elevation in plasma lipoproteins is hyperalphalipoproteinemia, characterized by elevated plasma levels of high-density lipoproteins. The elevation in HDL leads to slight increase in total plasma cholesterol values. Other plasma lipid components (LDL, VLDL, and triglycerides) are normal (Cooper *et al.*,1992*)*. The majority of cases of hyperalphalipoproteinemia are genetic with either a dominant or polygenic inheritance. Secondary elevations of HDL have been related to various factors such as weight reduction, regular exercise, moderate alcohol intake, estrogen administration, exposure to chlorinated hydrocarbon pesticides, and biliary cirrhosis. Patients with these conditions usually do not present any distinguishing clinical features. As previously mentioned, hyperalphalipoproteinemia is associated with a decreased risk of coronary atherosclerosis and with increased longevity (Brewer *et al.*,1983).

Hypoalphalipoproteinemias

Hypoalphalipoproteinemia probably is the most clinically significant hypolipoproteinemia in view of the fact that considerable evidence suggests that low levels of plasma HDL cholesterol are related to an increased incidence of coronary heart disease in high-risk populations.

According to the NIH Consensus Conference, the finding of HDL cholesterol values below 35 mg/dl

constitutes an independent risk factor for coronary artery disease. Several factors have been identified as causing a decrease in HDL cholesterol. These include ill-defined genetic factors, obesity, cigarette smoking, physical inactivity, hypertriglyceridemia, oral contraceptives, beta-adrenergic blocking drugs, thiazide diuretics, and cholesterol-reducing diets (Brown and Goldstein,1984).

Other hypolipoproteinemias comprise two rare disorders characterized by a decrease in the concentration of lipids in plasma and an autosomal recessive inheritance.

Abetalipoproteinernia

Usually appears early in childhood, and because of a defective production of apoprotein B, there is absence of chylomicrons, VLDL, and LDL in the plasma. The plasma cholesterol level is usually less than 75 mg/dl and that of triglycerides less than 15 mg/dl. The main clinical features are malabsorption of fats, peripheral neuropathy, ataxia, retinitis pigmentosa, and acanthocytosis (Castelli *et al.*,1977).

Tangier disease

Is a condition that also manifests in childhood and is characterized by the absence of HDL from the plasma. This defect leads to the production of abnormal chylomicron remnants, which are stored as cholesterol esters in cells of the phagocytic system. Levels of plasma cholesterol are usually less than 100 mg/dl and that of triglycerides range from 100 to 250 mg/dl. The main clinical features are enlarged orange tonsils, corneal opacities, and infiltration of the bone marrow and the intestinal mucosa. Patients with this illness are at increased risk for premature atherosclerosis (Goldstein *et al.*,1983).

Dyslipoproteinemia

Is the term utilized for conditions in which structurally abnormal lipoproteins circulate in plasma. Such a defect is seen in lecithin cholesterol acyltransferase (LCAT) deficiency. This is a rare disorder in which decreased activity of this enzyme leads to a large accumulation of unesterified cholesterol in plasma and body tissues. Laboratory findings include a variable level of total plasma cholesterol with decreased esterified cholesterol, an increase in unesterified cholesterol and increased VLDL. The structure of all the lipoproteins is abnormal. The condition usually presents in young adulthood with corneal opacities, renal insufficiency, hemolytic anemia, and premature atherosclerosis (Council on Scientific Affairs,1983).

Apoprotein CII Deficiency

Is a rare autosomal recessive disorder caused by absence of apoprotein CII, a required co-factor for the activity of lipoprotein lipase. The ensuing functional deficiency in this enzyme leads to a clinical picture similar to that described above for congenital lipoprotein lipase deficiency. However, in contrast to what occurs in the latter disorder, affected individuals are diagnosed at later age and rarely present eruptive xanthomas. The usual presentation is also with recurrent abdominal pains secondary to acute pancreatitis. At times the diagnosis is made by chance discovery of a milky serum.

Due to the inherent defect in this condition, in which lipoprotein lipase is not activated, both chylomicrons and VLDL are elevated in the blood causing a type 1 or type 5 lipoprotein pattern (Crook,1971).

Familial dysbetalipoproteinemia

Also called familial type 3 hyperlipoproteinemia, is a condition inherited through a single gene mechanism whose clinical presentation requires the presence of other genetic or environmental factors. Elevation of both plasma cholesterol and triglycerides occurs because of accumulation of remnant VLDL particles in the plasma. The metabolic defect in most patients occurs in apolipoprotein E. This has three common alleles, designated E^2, E^3, and E^4. Patients with this disorder have only apolipoprotein E_2 in VLDL, which is less effective in facilitating clearance of remnants than E_3 or E_4. The condition occurs only in individuals who are homozygous for E^2, that is, those with an E^2/E^2 genotype. Clinical evidence of hyperlipoproteinemia usually appears after the second decade. The characteristic clinical findings are xanthoma striata palmaris and tuberous and tuberoeruptive xanthomas over the elbows and knees. The disorder is associated with severe atherosclerosis of the coronary arteries, abdominal aorta, and peripheral arteries(Burtis *et al.*,2008).

The diagnosis is facilitated by encountering a broad beta band on lipoprotein electrophoresis. Confirmation can be obtained in specialized laboratories either by measuring the chemical composition of the VLDL fraction after ultracentrifugation of the plasma or by determining for the E^2 allele after isoelectric focusing of remnant proteins (Cooper *et al.*,1992).

Conclusion

Lipids have important roles in virtually all aspects of life, ranging from serving as hormones, energy source, aiding in digestion, to acting as structural components of the membranes of cells. All these good attributes do not undermine the fact that lipids are involved in the development of atherosclerosis, a pathogenic process that is the underlying cause of the common cardiovascular disorders such as, myocardial infarction, cerebrovascular disorders, and other peripheral vascular diseases. In other to ensure that these anomalies' are curtail, lipid has to be maintained physiologically, evenly distributed and transported in the body system which are the primary function of lipoproteins. As they are classified base on their heaviness and electrophoretic mobility; each has a role to play, either to take lipids off the cells or the other way round, the primary aim is to maintain lipid homeostasis. Determination of the quantity of lipid contents of these particles has a proof towards unveiling the state of the system with lipid availability. Different methods are employed in the investigation of the aforementioned in the laboratory.

Glycerol is a product of normal metabolic processes, it is present in serum. The measured quantity of TG in serum is slightly overestimated, if not corrected for endogenous glycerol. In healthy individuals, it represents the equivalent of < 10mg/dl (0.11mmol/l) of TG which is not significant clinically. In certain conditions like DM, emotional stress, i.v. drug administration of glycerol containing nutrients, contamination of blood collection devices, and prolong blood storage under refrigerator, this error becomes significant and could be corrected by "blanking out" as done by some laboratories or by consuming the glycerol content enzymatically in prereaction steps before measuring TG. The TG can also be corrected by a mathematical expression as done by some laboratories.

$$\text{Corrected TG} = \frac{OD\ of\ std}{3} \times 2$$

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